# Update on diagnosis, treatment and monitoring of diabetes in cats

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Diabetes mellitus is a common endocrine disease in cats. It is currently assumed that approximately 80% of diabetic cats suffer from a type 2 like diabetes, which is a heterogeneous disease due to a combination of insulin resistance and β-cell failure. Risk factors are obesity, increasing age, male gender, being neutered, physical inactivity, glucocorticoid and progestin administration, and being a Burmese cat (at least in some countries). Other specific types of diabetes (formerly called secondary diabetes) account for approximately 20% of cases and include pancreatitis, hyperadrenocorticism, hypersomatotropism and the application of progestins or glucocorticoids.
Diabetes typically occurs in middle-aged to old cats, with a strong sex predilection for males. Approximately 60% of diabetic cats are overweight, 35 are normalweight and 5% underweight (1). Most diabetic cats have the classical symptoms of diabetes, namely polyuria, polydipsia, polyphagia and weight loss. About 10% have overt symptoms of diabetic neuropathy, manifested as hind limb weakness, decreased ability to jump and plantigrade posture. Cats are prone to stress hyperglycemia which has to be differentiated from hyperglycemia due to diabetes by repeated blood glucose measurements or by measurement of fructosamine. Further work-up should clarify the severity of diabetes, presence of concurrent disease and presence of underlying disease/factors. Treatment should be initiated immediately and include twice daily insulin application (intermediate acting insulin) and high-protein-low carbohydrate diet (therapy will be covered in more detail in the presentation on “Diabetic remission”).
The general aim of therapy is to provide a good quality of life by eliminating clinical signs of diabetes and preventing complications such as hypoglycemia and ketoacidosis. Immediate therapy may reverse the effects of glucose toxicity and the diabetes may go into remission. Initially, frequent re-evaluations should be scheduled, with time, intervals can be extended. In our hospital re-evaluations are suggested 1, 3, 8, 12 weeks after diagnosis and then approximately every 4 months. The owner should assess his animal with regard to the clinical signs of diabetes mellitus on a daily basis. Body weight should be taken at least once a week. It is important that the owner is familiar with the clinical signs of the most important complications of diabetes mellitus, i.e. hypoglycemia and diabetic ketoacidosis. In general, single measurements are considered insufficient to assess metabolic control. Blood glucose curves (BGCs) are necessary to evaluate insulin efficacy, glucose nadir, duration of insulin effect, degree of fluctuation and the Somogyi effect. We prefer that owners give insulin and food at home, and then bring the animal quickly to the hospital (within 2 hours) for a BGC. This approach eliminates the effect of lack of food intake on blood glucose levels, at least in animals which are only fed at the time of insulin administration.
However, the procedure is time-consuming and expensive and therefore in many patients may not be carried out as often as indicated and the concentration of blood glucose can be markedly influenced by the stress of hospitalisation. We therefore recommend to the owners of diabetic cats to perform so-called home-monitoring

(HM) of capillary blood glucose and approximately 70% of the owners are willing and able to do this. Usually, we introduce HM 3 weeks after the initial presentation. Capillary blood is obtained from the pinna or paw and glucose is determined with a portable glucometer (PBGM). Owners can certainly be taught to interpret a BGC; however, we prefer that decisions regarding changes in the insulin dose be made by the veterinarian and therefore, BGCs be sent to the hospital. Ideally, the glucose concentration ranges from 12 – 15 mmol/l before the insulin injection with a nadir from 5 – 9 mmol/l. A low nadir may occur in insulin overdose (including sudden improvement of insulin-resistant states), excessive overlap of insulin actions and lack of food intake. If the nadir is > 9 mmol/l, insulin underdose, the counter-regulatory phase of the Somogyi effect and technical problems involving the injection of insulin by the owner must be considered. In diabetic pets that already receive high doses of insulin (> 1.5 U/kg/injection), concurrent diseases causing insulin resistance are also possible. The duration of effect is defined as the time from insulin injection through the glucose nadir until the blood glucose concentration exceeds 12 – 15 mmol/l. If the duration is less than 8 – 10 hours, the animal usually has signs of diabetes, and if the duration is longer than 14 hours and the insulin is given twice daily, the risk of hypoglycemia increases. In humans, it is well known that blood glucose concentrations can vary markedly from day to day. There is also a high degree of variability among BGCs of diabetic pets. One of the major advantages of HM is that it enables frequent generation of BGCs, which may be of particular importance in animals that are difficult to regulate. In those cases, more than one BGC can be generated at home before a change in treatment is initiated. HM has replaced measurement of urine glucose nearly completely in our hospital.
We routinely measure the fructosamine concentration during the re-evaluations. It increases when glycemic control worsens and decreases when glycemic control improves. Since even well-controlled pets may be slightly to moderately hyperglycemic throughout the day, fructosamine concentrations will not usually decrease into the normal range. On the contrary, a normal fructosamine concentration should raise concern about prolonged periods of hypoglycemia (insulin overdose, diabetic remission). Metabolic control is usually good when fructosamine levels are between 350 and 450 µmol/l, moderate when values are between 450 and 550 µmol/l and poor when levels are > 550 – 600 µmol/l (2).

1. Reusch CE, Robben JH, Kooistra HS. Endocrine pancreas. In: Rijnberk A, Kooistra HS. eds. Clinical Endocrinology of Dogs and Cats. 2nd ed. Hannover: Schlütersche Verlagsgesellschaft, 2010; 155-185.

2. Reusch CE: Feline diabetes mellitus. In: Ettinger SJ, Feldman EC. eds. Textbook of Veterinary Internal Medicine. 7th ed. St. Louis Missouri: Elsevier, 2010; 1796-1816.